ABRIDGED PRODUCT INFORMATION. INDICATIONS: 1. Prevention or treatment of shock associated with reduction in effective circulating blood volume due to: (a) haemorrhage (visible, concealed); (b) loss of plasma (burns, peritonitis, pancreatitis, crush injuries); (c) loss of water and electrolytes from persistent vomiting and diarrhoea. 2. As a plasma substitute in surgery where controlled haemodilution is employed. 3. Procedures involving extracorporeal circulation. 4. Carrier for insulin infusion. 5. Isolated organ perfusion. CONTRAINDICATIONS: Known hypersensitivity to constituents of Haemaccel. Existing anaphylactoid reactions. WARNINGS: Infuse clear solutions only. Once the bottle is opened, the solution should be used immediately. Any unused contents should be discarded, since Haemaccel contains no preservative. There is a residual air volume in the container and pressure infusions with the plastic infusion bottle must be carried out under supervision. Rapid infusion of Haemaccel, to normovolaemic patients, may stimulate release of histamine. Urticaria and rarely bronchospasm and hypotension may occur (see Adverse Re-actions). Administration of red cell intravascular/interstitial fluid volume may be hazardous. Blood losses up to 25% of the blood volume can be replaced by Haemaccel alone. Haemaccel may be given only after the prophylactic use of H1 and H2 receptor antagonists to the following patients. Patients with known allergic conditions such as asthma, a history of histamine response or patients who have received a histamine releasing drug (such as anaesthetics, muscle relaxants, analgesics, anticholinergic ganglion blockers) within 7 days prior to Haemaccel administration as they increase the risk of histamine release. USE IN PREGNANCY AND LACTATION: Haemaccel, for its usual indications, is not contra-indicated in pregnancy. However, particular care should be exercised when fluid or volume replacements are administered during or immediately after pregnancy. Haemaccel is employed during and following labour and no harmful effects on the newborn have been reported. It is not known whether polygeline is excreted in breast milk. ADVERSE EFFECTS: Transient skin reactions (urticaria, wheals), hypotension, tachycardia, bradycardia, nausea/vomiting, dysponea, rises in temperature and/or chills may occasionally occur. Rare cases of anaphylactoid reactions have been reported with bronchospasm, tachycardia and severe hypotension. Quincke's oedema has also been reported in such instances. These reactions are due to histamine release and may be the result of the cumulative effect of histamine-releasing drugs. They are not true anaphylactic reactions on an immunological basis. If side effects occur, the infusion should be discontinued immediately. If necessary, treatment should be given as follows: Mild reactions: administer corticosteroids and antihistamines. Severe reactions: if appropriate, immediately inject adrenaline (slowly I.V.), high doses of corticosteroid (slowly I.V.), volume replacement (e.g. human albumin, Ringer's lactate solution), oxygen and, if necessary, resuscitation. DOSAGE AND ADMINISTRATION: Haemaccel is administered intravenously, and can be infused immediately. Adults and children: 1. Prevention or treatment of shock associated with reduction in effective circulating blood volume due to: (i) haemorrhage, blood loss up to 1500 mL - correct by use of Haemaccel alone, blood loss in the range 1500-4000 mL - recommended ratio, Haemaccel/whole blood is 1:1, blood losses above 4000 mL - recommended ratio Haemaccel/whole blood is 1:2. The rate of infusion and total dose employed will be governed by clinical assessment. In acute situations of severe rapid blood loss, large volumes and rapid infusion may be required. The haematrocrit should not be permitted to fall below 25 to 30 volume % during therapy. (ii) relative hypovolaemia.Normovolaemia and a high speed of Haemaccel infusion are considered as factors contributing to anaphylactoid reactions in suceptible individuals. Where Haemaccel is used to restore circulating blood volume in the absence of loss of intravascular fluid, the patient should be carefully observed for skin reactions, difficulty in breathing or precipitous fall in blood pressure. (iii) burns. The management of extensive burns should be undertaken by specialised units. The volume of Haemaccel and crystalloid given should be varied according to the clinical response of the patient and the assessment of renal function. (iv) water and electrolytes. Haemaccel may be used to restore deficiencies in circulating blood volume in conditions such as persistent vomiting and diarrhoea. 2. As a plasma substitute in controlled haemodilution. Autologous blood transfusion and haemodilution techniques involve the collection of 2-3 units of blood from the patient after the induction of anaesthesia, each unit being simultaneously replaced by 500 mL of Haemaccel. During the operation, blood losses are immediately replaced with an equal volume of Haemaccel, as long as the haematocrit is above 0.25-0.30, or with blood alone when the haematocrit falls below this level. 3. Procedures involving extracorporeal circulation. Use for priming heart-lung machine, pump oxygenator, plasmapheresis and plasma exchange has been documented (see full Prescribing Information).4. Carrier solution for insulin infusion. Haemaccel, added to the infusion fluid before the addition of insulin, minimises adsorption of insulin onto glassware and plastic and allows a constant delivery rate to be maintained. Concentrations of polygeline as low as 0.5X (100 mL: Haemaccel with 500 mL infusion solution), are effective for this purpose. 5. Iso-lated organ perfusion. Use in animal models has been documented (see full Product Information). With impaired hepatic function - no modification necessary. With impaired renal function, Haemaccel has a beneficial effect on renal function and no exacerbation of pre-existing renal disease need be expected. COMPATIBILITY: Provided sterile precautions are observed, Haemaccel may be mixed with ordinary infusion fluids (saline, glucose, Ringer's solution, etc.) and with drugs acting on the cardiovascular system, corticosteroids, muscle relaxants, barbiturates, vitamins, streptokinase and antibiotics of the penicillin series, provided they are water soluble. Citrated blood (stored blood for transfusion) must not be mixed with Haemaccel (because the calcium ions in Haemaccel would cause reclassification). However, it may be transfused immediately before or after an infusion of Haemaccel. There is no objection to mixing heparinized blood with Haemaccel. In common with all infusion fluids, Haemaccel - for physiological reasons - should not be administered at a low temperature. PRESENTATION: Flexible plastic infusion bottle in 500 mL. REFERENCES: 1. Haemaccel Product Information. 2. Silvay, J et al Journal of Thoracic and Cardiovascular Surgery (1968), 55:350-358. 3. Merikallio, E, Annales Chirurgiae et Gynaecologiae (1976), 65: 138-144. 4. Donahue, J.G., et al NEJM (1992), 327:367. 5. Dax, E., et al, MJA (1992), 157:69. Hoechst Marion Roussel Australia Pty. Ltd. ACN 008 558 807, Private Mail Bag 2067, Lane Cove 2066. Further information available on request. Haemaccel® is a registered trademark of Hoechst AG.

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TRAUMA Grapevine

Contents:

Introduction

The article by Dr. Kolkman reviewing the management of blunt injury to the thoracic aorta raises a number of questions.

Should we now proceed to newer technology with spiral CT and rapid reconstruction, can we translate results from major trauma centers in the United States to major trauma services in Australia? Is special expertise required in trauma radiology? From attending meetings throughout Australasia, there are a number of anecdotal cases continually being discussed in relation to diagnostic error, both from angiography and CT scanning in relation to blunt aortic injury. Perhaps it is time for a national survey of the diagnostic accuracy of some of the key investigations we currently use for trauma care?

For those of you continually at the coal face in the resuscitation room and through the early part of definitive care - the concept of a Consortium Of Care for Urgent Priorities (C.O.C.U.P.) should be borne in mind. That is where the multi trauma patient with multi system involvement requires multiple procedures, potentially under multiple specialists with the plastic surgeon suturing the face, the orthopaedic surgeon applying the back slab, the facio maxillary surgeon fixing the mandible, the opthalmic surgeon performing the fundoscopy on the eye, the vascular surgeon reviewing the injured vessel, the neurosurgeon evacuating the haematoma and the urologist performing the nephrectomy. This exaggeration of the normal multi trauma injury scenario which can occur with resulting significant delays, multiple trips to the operating theatre and for those who believe in it, the concept of the multiple heat theory for activation of multiple organ failure. As specialisation increases we must not forget the patient and avoid the C.O.C.U.P.

Eddie Geller and his team at ISD are currently working on a major upgrade of our web page. We hope to bring you "Search the Site" facilities, new format for MCQ's, new format for Radiology of trauma cases. This upgrade will be on line in May.

Michael Sugrue

Blunt Trauma of the Thoracic Aorta

Karel A. Kolkman, Trauma Fellow, Liverpool Hospital

Background

Recently we saw a patient who was involved in a high speed frontal car crash with breaking of the steering wheel against his chest. There were no recorded episodes of hypotension. Clinically there was a central flail chest, chest X-ray showed a fractured right clavicle and some widening of the superior mediastinum, no pneumothorax. CT scanning of the chest revealed a large ventral left sided pneumothorax (50%) and a double density in the aortic arch with no obvious intimal flap or false aneurysm. There was a haematoma around the aortic arch. Arch angiography only showed some flow disturbance but no evidence of rupture. Trans Oesophageal Endosonography (TOE) showed a small thrombus in the most proximal part of the descending aorta, no disturbance of flow. The injury was considered to be not of clinical importance and the patient was not operated on. Repeat CT scanning two weeks later showed resolution of the haematoma, no flap or false aneurysm. Recent literature was reviewed to determine if there is any support for non-operative management of suspected aortic injuries and to update our diagnostic policies if necessary.

Biomechanics and pathology

An Australian study showed 53% of patients sustaining a Blunt Aortic Injury (BAI) were involved in a motor vehicle crash, 21% in a motorcycle crash, in 24% a pedestrian was hit by a vehicle and falls greater than 5 metres caused the aortic injury in 3%[1].

Three patient populations can be distinguished[2]. A large majority die at the scene. The second group is hemodynamically unstable on arrival or becomes unstable shortly after arrival and has only a 2% survival rate. The third group lives to undergo aortography and has a 75 % survival after appropriate management.

There are 4 theories describing the mechanism by which injury to the thoracic aorta occurs[2]: 1 "points of attachment" 2 "torsion" 3 "hydrostatic pressure" 4 "osseous pinch". The site of thoracic aortic injury is ascending aorta in 3%, proximal descending aorta in 73% and 24% at the level of the diaphragm. In aortic ruptures all three layers are torn and it is the surrounding tissue that limits the developing false aneurysm (eg. the parietal pleura in the case of a ruptured descending aorta).

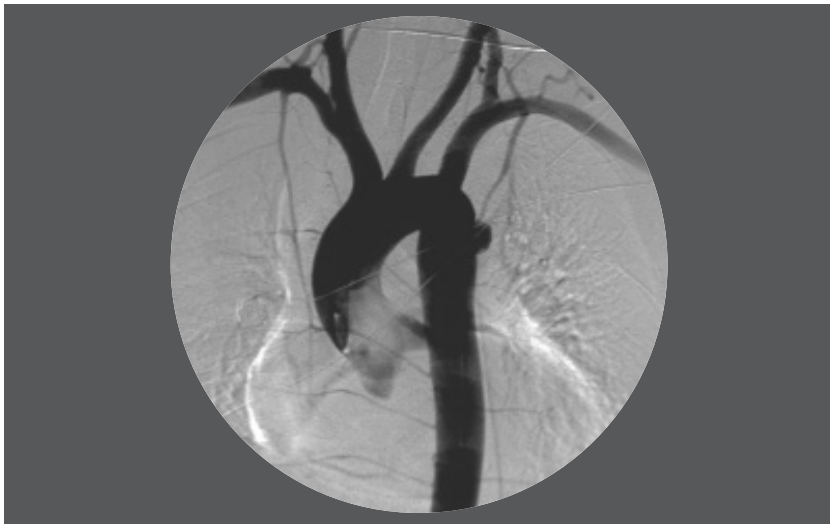
Screening

The best initial screening tool is the erect CXR[3]. Suggestive abnormalities are: 1 a widened mediastinum (more than 8 cm), 2 an obscure aortic knob, 3 depression of the left main stem bronchus, 4 right deviation of the nasogastric tube, 5 opacification of the aorto-pulmonary window, 6 widened paratracheal and paraspinous stripes and 7 apical capping. A normal CXR occurs in 8.3% of patients with a BAI. Therefore patients with significant deceleration or acceleration mechanisms should undergo a diagnostic test.

Continued next page

Blunt Trauma of the Thoracic Aorta

Continued



Diagnosis

Aortography is considered the gold standard diagnostic test for BAI. It is highly sensitive (67-100%) and specific (98-100%). Disadvantages are:

- it is an invasive procedure
- time consuming
- transport to the radiology suite
- use of contrast

False positive results have been reported in patients with atherosclerosis and in cases of a remnant of Botalli's duct (ductus arteriosem diverticulum).

Helical CT scanning is gaining more and more support. In a study of more than 6000 helical CT scans Pate et al[4] showed that no patient with a negative CT had a BAI. Subtle, indirect or indeterminate findings were followed by aortography. In an earlier study they reported a sensitivity of helical CT scanning of 100% (aortography 96.3%) and a specificity of 81.7% (aortography 96.3%). The advantage of helical CT scanning is that it reduces the need for aortography and can reduce the time the patient spends out of the resus room especially if they need a CT done for other injuries like a head injury. Disadvantages are:
no reliable imaging of the aortic branches and difficult imaging in obese patients.
For the helical CT scan to be really reliable there has to be strict protocol with 7mm sections and 3.5 mm reconstructions.

TOE can accurately image the aortic arch but is insufficient for the detection of injuries to the proximal ascending aorta and the aortic branches. TOE may pick up small intimal tears that are not seen by the two other modalities and these "minor" injuries are considered clinically insignificant by some (personal communication with Ken Mattox). Last but not least, TOE requires a dedicated and experienced cardiologist to perform the investigation at any time a trauma patient comes in.

Treatment

It has been stated that all blunt aortic injuries are full thickness ruptures through all 3 layers and therefore require urgent surgical treatment. In a select group of patients where better preparation or stabilisation before surgery is deemed necessary or to allow for orderly planning of surgery for other injuries, a selective delay of aortic repair can be safely accomplished. A medical management protocol to delay surgery, including administration of ß-blockers with or without vasodilators has been recently reported to be a safe alternative to urgent surgery[4] in selected cases. The target blood pressure was between 100 and 110 mmHg systolic.
Basically there are two options for surgical repair. Most surgeons (65%) will use some kind of by-pass technique while 35% of surgeons involved in a multicenter trial of the American Association for the Surgery of Trauma[5] will opt for a clamp and sew technique. In this study the overall mortality was 31% and paraplegia occurred in 8.7% of patients. Two factors were identified to increase the risk for paraplegia: clamp and sew technique, and aortic cross clamp time of more than 30 minutes.
At this stage there is inconclusive data to support non-operative management of aortic injuries seen on CT scan or aortography.

Conclusion

At Liverpool Hospital aortography is still the primary diagnostic tool for suspected BAI. I propose a change of management.
Based on what is stated above:
all patients with any suggestive abnormalities on the CXR will have aortography.
If there is a low grade suspicion (some widening of the mediastinum but less than 8 cm) or if the suspicion is only based on the mechanism of injury a helical CT scan will be done according to a new radiology protocol.
Where the helical CT scanning demonstrates a vascular abnormality or a haematoma in the mediastinum aortography will be performed.
All of this will be done in conjunction with the radiologist and his focussed report.
There will be no place for TOE in the emergency setting.
The hypothesis is that this change in policy will reduce the number of aortograms, reduce time spend outside the resus room and fewer movements of the patient from one table to the other.

The management of BAI is changing, with increasing recognition of the role of helical CT scanning. These have to be reported on by a specialist following a detailed protocol of scanning. International centres are increasingly using a more cautious approach to surgery with delayed surgery made possible by medical control of blood pressure.
Above all remember the possibility of Blunt Aortic Injury.

Suggested reading:

1. Hills et al.: Traumatic thoracic aortic rupture: investigation determines outcome. Aust NZ J Surg 1994; 64: 312-318
2. Mattox KL: Red river anthology. J Trauma 1997; 42: 353-368
3. Guidelines for the diagnosis and management of blunt aortic injury. www.east.org
4. Pate et al: Traumatic rupture of the aortic isthmus: program of selective management. World J Surg 1999; 23: 59-63
5. Fabian et al.: Prospective study of blunt aortic injury: multicenter trial of the AAST. J Trauma 1997; 42: 374-383

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LIVTRAUMA website:
http://www.swsahs.nsw.gov.au/livtrauma

LETTER TO THE EDITOR

Dear Editor,

I read with great interest Tim Skinner's excellent synopsis of hypothermia in severe trauma[1]. There are a couple of points that I would like to add. Recently, Krishna et al[2] suggested that the "reasons for the development of hypothermia and acidosis in the severely injured patient are perhaps not as obvious as it would seem". Although their study had limitations, and many possible hidden confounding factors, they found that hypothermia "was not strongly correlated with the time since the accident occurred or various therapies such as the administration of fluids". Instead, there "was the impression that hypothermia was more strongly associated with the extent and pattern of the injuries".

There was a provocative little animal study reported in 1996[3] which subjected rats to haemorrhage (30ml/kg), then one hour of shock, followed by 2:1 crystalloid blood resuscitation (60ml/kg) at ambient temperature. They were compared to a control group that underwent neither haemorrhage nor resuscitation. The temperature drop in the haemorrhage group was fastest during the hour of hypotensive shock. The rate of temperature change correlated with the shock phase and BP during the shock phase, but not with the resuscitation phase, or with the duration of shock or resuscitation. In fact, an increase in temperature was noted in just over half the rats during resuscitation. Could it be, as was proposed, that traumatic hypothermia is directly related to haemorrhage/hypotension rather than resuscitation? As Tim points out in his article, reduction in heat production is a feature of traumatic injury, and it may well be, as he suggests, that an increase in metabolic heat production may just not be possible due to inadequate tissue oxygenation.

Perhaps even more important is the major thrust of Krishna et al's[2] paper, that there may be a group of patients with severe truncal multitrauma that do better with "damage control" surgery versus conventional surgery, and that indicators such as base deficit, hypothermia and ISS may help identify such patients. As I am not a surgeon, Michael, I would be very interested in your own views regarding these techniques.

Despite these intriguing reports, it would seem likely that the origin of hypothermia in severe trauma is multifactorial, and its detrimental effects well documented. As Tim concludes, every effort should be made to prevent the development of hypothermia, and effective, rapid treatment may improve patient outcome.[1]

With kindest regards,
Craig Hore
Director of Emergency Services
Port Macquarie Base Hospital

References:

1. Skinner, T: Hypothermia and severe trauma. Trauma Grapevine 1998; 1 (14): 1-4
2. Krishna, G; Sleigh, JW; Rahman, H. Physiological predictors of death in exsanguinating trauma patients undergoing conventional trauma surgery. Aust NZ J Surg 1998; 68: 826-829.
3. Bergstein, JM; Slakey, DP; Wallace, JR; Gottleib, M. Traumatic hypothermia is related to hypotension, not resuscitation. Ann Emerg Med 1996; 27: 29-42.

REPLY

Hypothermia has two well known clinical effects:
to preserve life, and to kill.

Hippocrates advocated packing haemorrhaging patients in snow and ice[1]. Napoleon's chief battlefield surgeon, Barron de Larry, noted that injured patients, who were closest to the fire, usually were the first to die. And Basset remarked on the increased survival of injured soldiers left in the snow compared to those treated with warm blankets and heated drinks[2].

In response specifically to your question, that traumatic hypothermia is directly related to haemorrhage hypotension rather than resuscitation. This may be partly true. However, to optimise outcome there is no doubt among a number of studies perhaps the best is by Gentillo from Harper View Medical Centre in 1997[3]. In this study of 57 hypothermic patients rapid rewarming was undertaken in half the patients which were equally matched. Continuous arterial venous rewarming group required less resuscitation to the same haemodynamic goals and were significantly more likely to rewarm. Only 2 (7%) of the 29 patients who underwent CAVR failed to warm to 36°C and both died, whereas 12 (43%) of 28 patients who underwent standard resuscitation failed to reach 36°C and all 12 patients died. So I believe that resuscitation has an important part to bear in the outcome from hypothermia both in its contribution and in its treatment.

In relation to your second question, damage control is a very important issue and surgeons must bail out. They should be encouraged by their anaesthetic comrades to do this on occasion. There is no drill for fancy GI anastomosis and dissection in patients who have undergone major transfusion who are hypothermic and acidotic. Dr. Bill Schwab from Philadelphia will be discussing this very issue at SWAN VII in August. It has particular surgical implications, as surgeons are trained to be meticulous and the concept of bailing out of somebody's abdomen is aesthetically distasteful for technically brilliant surgeons. It is however, something we will see increasing amounts of both in the abdomen and in the chest in Australian trauma care.

References:

1. Adams F. The Genuine Works of Hippocrates. New York: William Wood; 1886.
2. Bazzett HC. The effect of heat on the blood volume and circulation. JAMA 1938; 111:1841-1845.
3. Gentilello LM et al. Is hypothermia in the victim of major trauma protective or harmful? Ann Surg 1997: 226: 439-449.

Meetings

SWAN VII

SWAN VII will be held on the 5th & 6th of August, 1999 at Liverpool Hospital bringing to you seven (7) of the world leaders in Trauma care from overseas.

Dr. Bill Schwab, Philadelphia, USA
Dr. Peggy Knudson, San Fransisco, USA
Dr. Ken Boffard, Johannesburg, Sth Africa
Dr. Don Trunkey, Oregan, USA
Jorie Klein, Dallas, USA
Lt. Col. Tim Hodgetts, Aldershot, UK
Dr. Don Jenkins, Texas, USA

This Traumaganza is a must for all Swanoholics!

Controversies in Civilian and Military Trauma

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Australasian Trauma Society Meeting

20th November 1999 Auckland 64 7 8383123

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For Sydney
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For Melbourne
peter.danne@nwhcn.org.au for

Case of the Month

A 20 year old motor vehicle passenger was involved in a high speed frontal collision at an intersection.

Pre-Hospital information

(M) Mechanism	Back seat passenger, wearing a seat belt. Alighted from vehicle unassisted.
(I) Injury	Seat belt abrasions on abdomen Pain in Lumbar spine Abdominal pain No clinical fractures
(S) Signs	Breathing unassisted, RR 24, Air entry R=L, Pulse 84/m, Systolic BP 120 mmHg GCS 15
(T) Treatment	Oxygen, C-Collar,

Resus Room:

Primary Survey

(A) Airway	Clear
(B) Breathing	Unassisted RR 15/m, SaO2 96% on 10L O2
(C) Circulation	Pulse 70/m, BP 74/47mmHg
(D) Disability	GCS 15

The team started immediate fluid resuscitation. 1 litre of saline over 1 hour was followed by 1 litre of Haemaccel. Analgesia was given, 25mg pethidine i.v.

Secondary Survey

This showed no injuries to his head and no neck tenderness on palpation. The patient's chest was also cleared.
Abdominal examination revealed seat belt marks from the right upper to the left lower quadrants. There was peri-umbilical tenderness but no guarding or rebound tenderness. Bowel sounds were present.
Examination of his spine showed a tender, boggy swelling at the level of L3/L4 with no neurological deficit.

Urgent x-rays of the cervical spine and chest showed no abnormalities. Spinal x-rays revealed a compression fracture of L2.

What would you do now? What are the potential diagnoses?

Review of last issue's case of the Month

Remember our 46 year old MB rider involved in a high speed accident.

Pre-Hospital Information

(M) Mechanism	Rider of motorbike
(I) Injury	Head, Chest, Right arms
(S) Signs	BP 90, P120/m, RR 34/min, GCS 12
(T) Treatment	Oxygen, C Collar, Haemaccel 750mls, Morphine 5mgs

Resus Room

Primary Survey and Early Management
On arrival the patient was in obvious distress.

Airway	-	intact
Breathing	-	oxygen saturation was poor on 92% RR32/m
Circulation	-	Pulse 130 per minute BP 110mmHg
Disability	-	GCS 13 Parasthesia R arm

The team organised urgent supine chest X-ray, C Spine and Pelvis X-rays. Breath sounds were reduced on the right side. Saturations came up to 96% on oxygen rebreathing mask.

The blood pressure at this point was 120 systolic.

Secondary Survey

His right humerus and right clavicle was clinically fractured. There was marked bruising over the right scapula. There was significant parasthesia over most of right arm - difficult to assess. His BP "stabilised" after 2L.
A chest x-ray revealed a widened mediastinum, a fractured 1st, 2nd and 3rd ribs on the right side, fractured clavicle, fractured humerus and fractured neck of scapula.

Where do you go from here?
Well a closer look at his right arm revealed a weak pulse. He proceeded to arch aortography and view of the innominate and subclavian artery. A decision was made not to do a chest CT as it would not have identified the subclavian and innominate with enough accuracy. The patient went straight to aortography . There was occlusion of the proximal subclavian artery and we proceeded to repair with interposition graft and exploration of the brachial plexus which were partly avulsed.

What are the most likely diagnoses?
Intimal flap in subclavian artery with associated brachial plexus injury.

Tracheal Intubation in Head Injury: A Practical Guide

Part 1. Why intubate? When to intubate and choice of drugs.

Cameron Hunt BSc, MB BS Anaesthetic Department, Liverpool Hospital
Stephen Fletcher MB BS, FRCA, MRCP Intensive Care Unit, Liverpool Hospital

Introduction

It is no surprise that the 'A' in the ABC of the primary survey represents the airway. The totally obstructed airway kills rapidly, as evidenced by the fact that of the 50% of trauma victims who die at the scene, airway obstruction is the cause of death in many. The partially obstructed airway leads to hypoxia and hypercarbia, both of which have a devastating effect on the traumatised brain.

Thus, airway patency in traumatic brain injury (TBI) is vital. How should airway patency be ensured in TBI? Few would doubt that in severe TBI, tracheal intubation is the gold standard for airway management, but when and how should this be achieved? Given the potential for intubation to cause further harm, the answer is not always easy. The first part of this article discusses the reasons why tracheal intubation is of benefit in head injury and provides guidance on selection of drugs for intubation. The second part gives practical advice on the conduct of intubation.

Tracheal intubation in head injury- The Pros

Trauma causes the primary brain injury, which may be perfectly survivable. The role of the trauma team is to identify pathology correctable by neurosurgery and prevent the secondary brain injury which worsens neurological outcome (Table 1). [1,2] Tracheal intubation relieves airway obstruction, permits controlled artificial ventilation, allows the delivery of a high-inspired concentration of oxygen and facilitates tracheal suction. Hypoxia, a potent cause of secondary brain injury, can be corrected and hypercarbia which causes cerebral vasodilatation (and increased intracranial pressure) is avoided. Where raised intracranial pressure is present or suspected, controlled ventilation and/or sedation (to reduce cerebral metabolic rate and thus intracranial pressure), are usually required. In TBI both of these interventions mandate tracheal intubation and clearly should not be undertaken without expert advice.

The indications for tracheal intubation are given in Table 2. There are times when a patient has a GCS greater than eight but is combative due to brain injury, hypoxia, drugs, pain and other injuries. Intubation allows these patients to be properly assessed and treated, reduces secondary brain injury and prevents self injury and injury to trauma team members.

Table 2.

INDICATIONS FOR TRACHEAL INTUBATION IN HEAD INJURY

- Airway compromise (failure of patency/risk of aspiration)
- Ventilatory failure or failure to oxygenate
- GCS 8 or less
- Warranted because of other injury (e.g. severe chest trauma)
- To facilitate management (e.g. CT scanning)

Table 1.

SECONDARY NEUROLOGICAL INSULTS IN TRAUMATIC BRAIN INJURY

(1) Hypoxia
(2) Low cerebral perfusion pressure because of hypotension
- Hypovolaemia
- Anaesthetic drug effect
- Other causes, e.g. tension pneumothorax, positive pressure ventilation

(3) Low cerebral perfusion pressure because of raised ICP
- Cerebral vasodilatation: Hypercarbia, hypoxia, hypotension
- Obstruction to cerebral venous drainage
- Coughing and straining
- Mass effect from intracranial bleeding

(4) Fitting
(5) Hypo/hyperglycaemia
(6) Hyperthermia
(7) Central nervous system infection

Tracheal intubation in head injury- The Pitfalls

Done properly, tracheal intubation can save lives. Done improperly, the complications associated with intubation can kill. There is potential for harm at every step:

- Anaesthesia is usually required. All anaesthetic agents depress blood pressure. This reduces cerebral perfusion pressure (CPP; mean arterial pressure minus intracranial pressure). A figure of 70 mmHg for CPP exists, below which cerebral hypoperfusion and secondary injury is thought to occur. [3] Although anaesthetics generally reduce the cerebral metabolic rate and therefore intracranial pressure, general anaesthesia does not protect against secondary brain injury caused by hypotension. [4]
- Hypoventilation and apnoea is associated with the use of all anaesthetic drugs. Apnoea is a desired effect of muscle relaxants. During the period between onset of drug effect and securing the airway with effective ventilation, this may lead to profound hypoxia and hypercarbia. If the patient can not be intubated, subsequent failure to provide effective ventilation and oxygenation will be catastrophic.
- The process of intubation may be traumatic to the structures of the face, mouth, pharynx, larynx and trachea. Intubation usually occurs before cervical spine injury has been excluded and exacerbation or production of a neurological deficit is possible.
- Oesophageal intubation happens even to the most experienced operators and can be difficult to detect quickly in the stressful trauma situation.

Tracheal Intubation in Head Injury: A Practical Guide (continued)

- Accidental extubation, bronchial intubation, disconnection of breathing systems and tube blockage are all possible.
- Sympathetic stimulation during intubation may lead to rises in ICP. In selecting the dose of anaesthetic agent, the balance between cardiovascular depression with overdosage and effects on ICP with underdosage must be considered.
- If anaesthesia is inadequate (and/or muscle relaxants are not used), laryngospasm, bronchospasm and coughing can occur.
- Obstruction to cerebral venous drainage will occur if tapes are fastened too tightly around the neck.
- Positive pressure ventilation, by increasing the mean intra-thoracic pressure may impede cerebral venous drainage and increase ICP.

Tracheal intubation in head injury - the balanced view

Given all of the above, the trauma team must clearly take a cautious view when considering intubation. In general however, if there is any doubt about the ability of the patient to maintain a clear airway, to breathe enough to maintain normocarbia and to oxygenate the arterial blood then intubation should be undertaken. Difficulty occurs when presented with the restless or combative patient, with respect to intubation solely to facilitate computerised tomographic scanning of the head. In this situation, guidance must be sought from the neurosurgeons. They may not think that scanning is appropriate; clearly then intubation may not be appropriate.

Tracheal intubation in head injury - Drug choices

It should always be assumed that the patient with severe TBI (GCS less than 9) has raised intracranial pressure with reduced intracranial compliance. Any increase in cerebral blood volume (due to hypercarbia, hypoxia, sympathetic stimulation, hypotension, cerebral venous obstruction, coughing, straining and excessive intrathoracic pressure) may lead to pronounced increases in ICP. As stated above, this will reduce the cerebral perfusion pressure and worsen outcome. The choice of anaesthetic, the technique of intubation and the method of ventilation can influence all these variables and thus the outcome of the procedure.

Anaesthesia

The skilled operator, presented with an extremely obtunded patient (GCS 3) may opt not to use any from of anaesthesia or muscle relaxation. This approach has its merits (cardiovascular stability and maintenance of spontaneous respiration), but anaesthesia is clearly necessary in patients with higher levels of consciousness. For the less skilled operator, anaesthesia and muscle relaxation are desirable in order to improve intubating conditions and the chance of success. An overview of the commonly used drugs is presented with particular emphasis on their effects on the cardiovascular system and ICP.

Thiopentone

Thiopentone is one of the most commonly used intravenous anaesthetics. It produces hypotension primarily by venous dilation and a reduction in venous return. In the hypovolaemic trauma patient this hypotension may be profound. Other problems with thiopentone include severe distal ischaemia if accidentally injected into an artery, dose dependent respiratory depression and a small but definite incidence of anaphylaxis.

Potential advantages with the use of a barbiturate in the head-injured patient include a decrease in cerebral metabolic rate and hence reduced cerebral blood volume and ICP. However, this beneficial reduction in ICP is negated if there is a greater decrease in cerebral perfusion pressure. As stated above, anaesthesia does not protect against the adverse effects of hypotension. [4]

Dose selection is a difficult issue. In the trauma situation, the patient may have a reduced requirement because of brain injury or presence of drugs or alcohol and reduced tolerance to the cardiovascular effects of the drug due to uncorrected hypovolaemia. In elective anaesthesia, titration of the drug against effect is possible. In the trauma setting, however, a 'rapid sequence' induction is mandatory. This requires as short a time as possible between the onset of anaesthesia and securing the airway, and careful titration of anaesthetic is not possible. Thus it is difficult to provide guidelines as to drug dosage; this applies to all anaesthetic agents. The 'normal'

"Difficulty occurs when presented with the restless or combative patient..."

doses of the commonly used agents are given in Table 3. Give less according to the conscious level and degree of cardiovascular compromise!

Propofol

This is an intravenous induction agent with a greater propensity than thiopentone to produce apnoea and depression of airway reflexes. Like thiopentone it reduces ICP by decreasing cerebral metabolic rate. However in the context of trauma and hypovolaemia it produces an arguably greater decrease in blood pressure than thiopentone. Propofol by infusion has gained widespread acceptance as the post-intubation sedative agent of choice.

Ketamine

Ketamine produces 'dissociative' anaesthesia, a state of profound analgesia, anaesthesia and amnesia where patients may appear to be awake, keeping their eyes open and hopefully maintaining airway tone. A major disadvantage of ketamine in the head-injured patient is a potential increase in ICP, although this effect is negated by neuromuscular blockade and controlled ventilation.

Ketamine increases sympathetic activity, but blood pressure is not necessarily maintained in the severely shocked. Availability and familiarity with ketamine are its main limitations.

Midazolam

Midazolam is the most frequently used benzodiazepine in anaesthetic practice. It has amnesic, sedative, anticonvulsant and relaxant properties. At doses of 0.05 mg/kg it provides the above properties as an aid to intubation. At doses of 0.1 - 0.2 mg/kg it will itself induce anaesthesia, although there is a ceiling to its anaesthetic properties; use of midazolam even in the larger doses does not guarantee anaesthesia. Comparatively, barbiturates and propofol have a greater effect on cerebral metabolic rate and ICP.

Opiates

If the patient is anaesthetised then, by definition, the patient will not experience pain. The use, therefore, of opiates during intubation in head injury is debatable. They will act as 'co-anaesthetics' and reduce

the required dose of other drugs, but whether this improves cardiovascular stability is again debatable. Opiates are useful as part of a sedative regimen following intubation.

Muscle relaxants

Suxamethonium

Suxamethonium produces neuromuscular blockade by prolonged depolarisation of the motor end plate at the neuromuscular junction. It is best stored at 4°C but will still retain most activity after several hours in solution at room temperature. It is unstable if mixed with alkali solutions such as thiopentone.

Onset of action is usually half to one minute following intravenous administration as evidenced by muscle fasciculation. Good intubating conditions are present within one minute of administration, although in the shocked patient with a prolonged circulation time, this may be much longer. Although considered short acting, the half life of suxamethonium is estimated at 2-4 minutes and the time to full recovery of muscle strength as long as 10 minutes.

The systemic effects and side effects of suxamethonium are numerous. Cardiovascular effects include bradycardia, particularly in children or with repeated doses. Cardiac arrest following suxamethonium may be due to hyperkalaemia (see below), anaphylaxis, or as a result of hypoxia.

The serum potassium normally increases by 0.5mmol/L with suxamethonium use, but in some conditions a much more exaggerated increase occurs. These include denervation syndromes (classically cord injuries), significant burns, extensive trauma e.g. crush syndromes and prolonged immobility. Hyperkalaemic arrest has also occurred in hypovolaemic acidosis. The exact risk period for these various conditions is debated. In the context of trauma, where the patient is likely to have been previously healthy, it is sufficient to bear in mind the possibility of hyperkalaemia if cardiac arrest occurs.

Opiates are useful as part of a sedative regimen following intubation.

In head injured patients a 'textbook' concern with suxamethonium use is an increase in intracranial pressure. Recent work has disputed this and the issue can be safely ignored.

A rise in intragastric pressure is probably a real phenomenon and emphasises the need for adequate cricoid pressure during intubation.

Intraocular pressure can double due to the effect of suxamethonium on the extraocular muscles. It is therefore relatively contraindicated in open eye injuries, although the well-being of the whole patient clearly takes precedence.

Other effects of suxamethonium are muscle pain which may last for days, prolonged neuromuscular blockade due to deficiencies of plasma cholinesterase and anaphylaxis and malignant hyperthermia which are rare but potentially lethal side effects. The incidence of malignant hyperthermia is highest in children (as high as 1 in 15,000).

Non-depolarising neuromuscular blocking drugs

The commonly used drugs are vecuronium, atracurium, cisatracurium, rocuronium, pancuronium and mivacurium. They produce muscle relaxation without causing fasciculation, muscle pain, hyperkalaemia, raised intraocular pressure, raised ICP or raised intragastric pressure and do not induce malignant hyperthermia. Thus they would seem ideal in the head-injured patient but for two considerations. Firstly, their duration of action following standard intubating doses is long, ranging from 10 to 20 minutes for mivacurium to 90 to 120 minutes for pancuronium. This means that in the event of failure to intubate and difficulty with ventilation, spontaneous breathing will not reappear for a long time. The anaesthetic teaching is that if a difficult intubation is predicted then do not use relaxants, especially non-depolarising relaxants.

Secondly, the time to intubating conditions is two to three minutes. This is long enough for profound desaturation in the presence of lung pathology, extremes of age and obesity. Clearly, once the patient is intubated, these cease to become concerns unless there is unplanned extubation.

Table 3.

NORMAL DOSES OF COMMONLY USED AGENTS

- Thiopentone 3-5 mg/kg
- Propofol 1-3 mg/kg
- Suxamethonium 1-2 mg/kg
- Fentanyl 2-5 (g/kg
- Morphine 0.1 mg/kg
- Vecuronium 0.1 mg/kg
- Propofol infusion 1-5 mg/kg/hr

Drug choice - A balanced view

The best course of action when confronted with the foregoing description of the pros and cons is to use the agents with which you are most familiar. In general, keep it simple and avoid combinations of multiple anaesthetic agents. Thiopentone and suxamethonium will not let you down. If hypotension occurs, treat it aggressively.

Vasopressors counteract the effects of anaesthetics on the circulation and it is appropriate to use them in the first instance.[2] Boluses of metaraminol (aramine; 100 to 250 µg boluses) or even adrenaline (50 to 100 µg boluses; 0.5 to 1 ml 1 in 10,000) are effective. If we assume that intracranial hypertension already exists (say an ICP of 20 cmH_2O) then the mean pressure should be 90 mmHg or above to ensure a CPP of at least 70 mmHg.

Conclusion

The primary brain injury is predetermined when the patient is wheeled into the emergency department. The task of the trauma team is to prevent secondary brain injury, particularly avoiding hypotension and hypoxia, which are independent predictors of a worse neurological outcome. Tracheal intubation helps prevent hypoxia, but must be conducted in such a way as to avoid hypotension.

The second part of this article will provide a practical approach to tracheal intubation and what to do when things go wrong.

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References:

[1] Wald SL, Shackford SR, Fenwick J. The effect of secondary insults on mortality and long-term disability after severe head injury in a rural region without a trauma system. J Trauma. 1993;34:377-381

[2] Chestnut RM, Marshall LF, Klauber MR et al. The role of secondary brain injury in determining outcome from severe head injury. J Trauma 1993;34:216-222

[3] Chestnut RM. Guidelines for the management of severe head injury: What we know and what we think we know. J Trauma 1997;42:S19-S22

[4] Pietropaoli JA, Rogers FB, Shackford SR, Wald SL, Schmoker JD, Zhuang J. The deleterious effect of intraoperative hypotension on outcome in patients with severe head injury. J Trauma 1992;33:403-407